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Washington, D.C. 20530

March 19, 1985

Dr. James K. Selkirk
Biology Division
Oak Ridge National Laboratory
P.O. Box Y
Oak Ridge, Tennessee 37830

Re: United States v. Reilly Tar & Chemical Corp.
no. 4-80-469 (D. Minn.)

Dear Jim:

I am sending you a copy of Dr. Eula Bingham's report. I have already arranged for a copy of the Wogan study on fluoranthene to be sent to you. Please tell me when you have designated your exhibits. I would also like to meet with you sometime in the next month to discuss your direct testimony.

Thanks for all your help.

Sincerely yours,

Assistant Attorney General
Land and Natural Resources Division

David Hird

By: David Hird, Attorney
Environmental Enforcement Section

Enclosure

cc: Dennis M. Coyne
Robert Leininger
William Sierks
Gordon Stoner
Jonathan Fleuchas
Paul Bitter

Statement of Eula Bingham

regarding St. Louis Park, Minnesota

Scientific evidence from animal studies and epidemiological investigations has accumulated over many decades that indicates coal tars and certain fractions derived therefrom are carcinogenic. Much of this work has been summarized by the International Agency for Research on Cancer. Coal tar, produced as a byproduct from coke ovens, has been tested under various conditions and is considered by scientific experts worldwide to be carcinogenic.

Distillation of coal tar produces fractions containing certain combinations of compounds contained in the original tar. Cresote is a fraction collected between 200-450^{o}C and contains a diverse group of compounds including phenols, cresols, polycyclic aromatic hydrocarbons and heterocyclic compounds. Numerous animal experiments have demonstrated that creosotes (or fractions thereof) are carcinogenic.

In addition to the carcinogenic effects of coal tar and cresote, there is evidence mainly from experimental studies that fractions of these two complex mixtures are cocarcinogenic. Phenolic compounds are present in coal tar fractions such as anthracene oil and creosote oil in high concentrations. Animal experiments reveal that phenols may act as cocarcinogens. According to the Tye et.al. squamous metaplasia and adenocarcinomas of the respiratory tract were seen more frequently in mice treated (inhalation) with coal tar containing phenols as compared with mice treated with coal tars

having the phenols removed. (References 1-11)

Many of the individual chemical carcinogens found in the complex mixture coal tar have been studied extensively but few as intensively as benzo(a)pyrene. One of the most significant aspects of this particular carcinogen has been its use as an indicator compound in complex mixtures. While it is fair to say scientists and public health officials may have missed other important chemical carcinogens and cocarcinogens in complex mixtures by focusing so much attention on benzo(a)pyrene, it is clear why this has happened when one examines the experimental data on this compound. Benzo(a)pyrene is a demonstrated carcinogen for many species of animals by practically all routes of administration and has produced tumors in multiple target organs. This compound also can be promoted or enhanced (cocarcinogenesis) by many non-carcinogenic chemical compounds. (References 12-15)

Also noteworthy in assessing the risks of this complex mixture of chemicals found in the wells is the demonstration of carcinogenic and/or cocarcinogenic potential of a number of specific chemicals. For example, pyrene and fluoranthene have been reported to be cocarcinogens.

Experience with complex mixtures (especially those derived from fossil fuels) provides evidence that even though various combinations of chemicals are present, one can be practically certain that a mixture containing certain carcinogens e.g. benzo(a)pyrene, dibenzanthracene, benzanthracene will produce cancers in an animal bioassay. While it is true that on a few occasions inhibitory activity has been demonstrated so that the

predicted carcinogenic activity, based on the concentrations of two or three carcinogens may be lower than expected, the vast majority of the time chemical carcinogens are at least additive and one can demonstrate synergistic activity (cocarcinogenesis) in many instances. (References 15, 19, 20, 21)

Experiments in the literature demonstrating the inhibitory effect of a single carcinogenic PAH when combined with one other carcinogenic PAH e.g. chrysene and benzo(a)pyrene may be very misleading in estimating the risks from the contaminated water (22). Most likely both chrysene and benzo(a)pyrene compete for the same enzymes that metabolize them to the active carcinogen so that in this particular experiment the full potential of the active carcinogen B(a)P was not expressed. However, chrysene while not possessing as much potency as B(a)P, clearly has been demonstrated to be an "initiator" of the carcinogenic process when followed by treatment with known promoters or cocarcinogens (IARC Monograph on Evaluation of the Carcinogenic Risk of Chemicals to Humans vol. 32, 1983). Thus, a false sense of security could emerge unless one remembers that chyrsene is an initiator and many compounds in the complex mixtures dissolved out of the coal tar and creosote fractions into the water supply are cocarcinogens. Isolated instances of so-called inhibition (usually binary mixtures) can be misleading when the real life situation is one where a complex multiple chemical mixture exists.

In general, human populations may be exposed to many carcinogens, such as polycyclic aromatic hydrocarbons, aromatic amines, solvents, etc. Most of these exposures to human population are involuntary resulting from

contamination of air, food, or water. While it may be possible for a few persons in a community to avoid these exposures to some extent, for example, by buying bottled water or specially organically grown foods, the vast majority cannot afford to do so. With the widespread distribution and use of chemicals in commerce it is not unusual to read reports of leaking tankcars of chemicals that pass through neighborhoods. Occupational exposures while not viewed as involuntary may actually be if the employee is not informed of the carcinogen in the workplace or cannot find employment in another job.

So-called "lifestyle" habits such as smoking and eating foods prepared in a manner so they are likely to contain carcinogens are voluntary and these "lifestyles" are often used as the justification to allow more and more involuntary exposures.

The scientific evidence indicates that all carcinogen exposures probably count and populations carry a "carcinogenic chemical burden." Several studies have influenced greatly this conclusion by the writer particularly those by Saffiotti and coworkers, Selikoff, and Bingham and Falk. (See Figures 1 and 2)

These studies (Selikoff) demonstrate that exposures to carcinogens and cocarcinogens may be multiplicative (Figure 1) in human populations and from animal studies it may be seen that cocarcinogen can enhance the effects of even a small quantity of carcinogen. In the study by Bingham and Falk a thousand-fold enhancement was observed. (Figure 2) In my opinion, one of the most disturbing animal experiments in its implications for human health is one by the Saffiotti group at the National Cancer Institute. In an experiment where intratracheal treatment with

benzo(a)pyrene adsorbed on a particle was given hamsters in a dosage calculated to produce a very low or no tumor response, no squamous cell carcinomas of the tracheobronchus resulted. When another type of carcinogen, diethylnitrosamine, was given at low doses subcutaneously to hamsters no tumors resulted. But when the diethylnitrosamine treatment was administered to hamsters that received the low doses of B(a)P a significant incidence of squamous cell carcinomas of the tracheobronchus was induced 31%. While one cannot extrapolate this directly to a human population by invoking mathematical models, it does suggest that there are severe biological consequences of very low doses of carcinogens even when they are different types of molecules.

Prudent public health policy should encourage the education of populations to better "lifestyle" habits i.e. avoid smoking (active and passive) and avoid foods containing known carcinogens. This rationale has meant that we as a nation have sought to keep manufacturers from adding carcinogens to food and to keep water supplies free from carcinogens. Substantial attention has been directed by the Federal government and certain state governments regarding the safety of food and water. Of special concern has been the introduction of contaminants with carcinogenic potential into the food and water supplies. Over and over it has been reiterated that one cannot determine individual thresholds for carcinogens so as to set a "safe" level of exposure of human populations to carcinogens. (Reference 23) In summary, residues present in St. Louis Park are rich in chemicals known to be toxic including carcinogenic, cocarcinogenic, and mutagenic potentials,

and the residues are likely to provide a continuing source of contamination for years to come. It is my opinion that steps should be taken immediately to clean up the area and water so that the population will not be exposed to more "coal tar derived" chemicals than populations living in areas without such a tar plant.

Figure 1

AGE STANDARDIZED LUNG DEATH RATES* FOR CIGARETTE SMOKING AND/OR OCCUPATIONAL EXPOSURE TO ASBESTOS DUST COMPARED WITH NO SMOKING AND NO OCCUPATIONAL EXPOSURE TO ASBESTOS DUST

Group	Exposure to Asbestos?	History Cigarette Smoking	Death Rate	Mortality Difference	Mortality Ratio
Control	No	No	11.3	0.0	1.00
Asbestos workers	Yes	No	58.4	+47.1	5.17
Control	No	Yes	122.6	+111.3	10.85
Asbestos workers	Yes	Yes	601.6	+590.3±	53.24

*Rate per 100,000 man-years standardized for age on the distribution of the man-years of 12,051 asbestos workers followed prospectively 1967–1976. Controls included 73,783 like men in a prospective study at the American Cancer Society for the same decade. Number of lung cancer deaths based on death certificate information.

Figure 2

Table 1.—*The Results of Repeated Cutaneous Applications of Low Concentrations of Benzo[a]pyrene in Decalin or n-Dodecane and Decalin*

Solution*	Original No. of Mice	No. Alive at 50 Weeks	Av Latent Period (weeks)	No. of Mice Developing Tumors: Malignant	No. of Mice Developing Tumors: Benign	Incidence of Tumors (%)
0.02% benzo[a]pyrene in decalin	20	12	58	5	1	50
0.02% benzo[a]pyrene in *n*-dodecane & decalin†	20	16‡	25	10	5	93
0.002% benzo[a]pyrene in decalin	30	20	...	0	0	0
0.002% benzo[a]pyrene in *n*-dodecane & decalin	30	26‡	48	7	2	35
0.0002% benzo[a]pyrene in decalin	30	21	...	0	0	0
0.0002% benzo[a]pyrene in *n*-dodecane & decalin	30	23	64	3	3	26
0.00002% benzo[a]pyrene in decalin	30	18	...	0	0	0
0.00002% benzo[a]pyrene in *n*-dodecane & decalin	30	24	80	5	0	21
0.000002% benzo[a]pyrene in *n*-dodecane & decalin	30	24	...	0	0	0
n-dodecane & decalin	40	30	...	0	0	0

* Dosage, 50 mg three times per week.
† *n*-dodecane and decalin, 50:50 by weight.
‡ Number alive after 50 weeks is given for those groups not developing tumors prior to 1 year of treatment. Final effective number (FEN) is given for the two groups that developed a substantial number of tumors before 1 year. The FEN is the number of mice alive at the median time of the occurrence of tumors plus any of those that had died previously with a tumor.

Table 2.—*The Carcinogenic Potency of Benz[a]anthracene in n-Dodecane or Toluene*

Solution*	Original No. of Mice	No. Alive at 50 Weeks	Av Latent Period (weeks)	No. of Mice Developing Tumors: Malignant	No. of Mice Developing Tumors: Benign	Incidence of Tumors (%)
1.0% benz[a]anthracene in *n*-dodecane	30	22†	42	16	1	...
1.0% benz[a]anthracene in toluene	40	29	76	5	3	...
0.2% benz[a]anthracene in *n*-dodecane	40	21	61	7	4	...
0.2% benz[a]anthracene in toluene	40	32	88	3	0	...
0.02% benz[a]anthracene in *n*-dodecane	40	20	69	4	0	...
0.02% benz[a]anthracene in toluene	40	18	67	1	0	...
0.002% benz[a]anthracene in *n*-dodecane	50	21	69	4	4	
0.002% benz[a]anthracene in toluene	40	32	...	0	0	
0.0002% benz[a]anthracene in *n*-dodecane	50	31	77	2	2	

* Dosage, 50 mg three times per week.
† Final effective number.

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